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Findings of 2-fluoro-2-deoxy-d-glucose positron emission tomography in hemorrhoids

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Abstract

Background: Hemorrhoids are very common in adults. The data regarding the incidence of high 2-fluoro-2-deoxy-d-glucose (FDG) uptake in hemorrhoids is incomplete. In this study, we evaluated FDG uptake in hemorrhoids and calculated the rate of high FDG uptake in these lesions.

Methods: One hundred and seventy six subjects who undertook whole body FDG–PET for health screening examination were investigated retrospectively. All patients had colonoscopy and 156 subjects were found to have hemorrhoids and 20 had no hemorrhoids. Quantitative analysis of FDG uptake in the anal region was performed by calculating the maximum standard uptake value (SUV_{max}).

Results: The SUV_{max} ranged from 1.8 to 4.1 (2.8 ± 0.6) for normal subjects and ranged from 1.4 to 8.3 (2.9 ± 0.8) for patients with hemorrhoids. No statistical difference was noted between these two groups using a Student's *t*-tests. If the highest SUV_{max}, which was 4.1 in normal subjects, was used as a cutoff, 5.1% (8/156) hemorrhoid patients had a SUV_{max} greater than 4.1.

Conclusion: Hemorrhoids can be one possible cause of focal high FDG uptake in the rectum.

Key words: 2-Fluoro-2-deoxy-d-glucose (FDG)—Positron emission tomography (PET)—Hemorrhoid—Maximum standard uptake value (SUV_{max})—Health screening examination

Hemorrhoids are one of the most common conditions and they frequently affect the adult population. Over half of the population has hemorrhoids by age 50. Hemorrhoids are swollen blood vessels around the anus or lower rectum. Thrombosis, inflammation and neovascularization are usually associated with the swollen blood vessels [1]. High fluorodeoxyglucose (FDG) uptake has been documented with thrombotic lesions, inflammation and neovascularization [2–5]. Therefore, a high FDG uptake in an inflamed hemorrhoid is possible. In 2006, Basu et al. [6] reported avid ^{18}F-FDG uptake in rectal hemorrhoids in a patient with metastatic medullar carcinoma of the thyroid. He assumed intense inflammation and hyperemia associated with the hemorrhoid might be responsible for avid FDG uptake. In the same year, Lu et al. reported another case of hemorrhoids with high FDG uptake [7]. To our surprise, these are the only two cases which have been reported with high FDG uptake in hemorrhoid patients. We are curious of the incidence of high FDG uptake in hemorrhoids?

The purpose of this study was to investigate FDG uptake in hemorrhoids and to realize the incidence of high FDG uptake in these lesions.

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Materials and methods

A total of 487 charts of healthy people, referred from the department of family medicine, examined by whole body FDG positron emission tomography (PET) for health screening examination from January 2007 to December 2009, were reviewed retrospectively. One hundred and seventy six subjects (71 females and 105 men, age ranging between 29 and 80 with an average of 53.2) undertook coloscopy within 3 months. Of the 176 subjects with reports of coloscopy, 20 had no evidence of hemorrhoids and 156 were found to have hemorrhoids including three external hemorrhoids, 149 internal hemorrhoids and four mixed type of hemorrhoids. This study was approved by the ethics committee of our hospital (DMR-99-IRB-010).

PET imaging

The PET studies were performed using Advance NXi PET scanner (General Electric Medical Systems, Milwaukee, WI) 40 min to 1 h after the intravenous injection of 370 MBq (10 mCi) of ^{18}F-FDG. The serum glucose levels of all subjects were checked to ensure the readings were less than 150 mg/dL. Before PET scanning, patients were encouraged to void to minimize activity in the bladder due to renal excretion of ^{18}F-FDG. The scanning was performed from the head to the upper thigh in 2D mode 4 min per bed position. Transmission scans were acquired with 68Ge rod sources for attenuation correction. Reconstruction of both transmission and emission scans used ordered-subset expectation maximization. The images were reconstructed and displayed in 3D and axial, sagittal and coronal reconstructions for interpretation. Quantitative analysis of ^{18}F-FDG uptake in the anal region was performed and maximum standard uptake value (SUV_{max}) was calculated. The highest SUV_{max} of the normal subjects was used as a cutoff value.

Statistical analysis

Statistical analysis of the results was made with the Statistica for Windows Release 4.5 package (StatSoft, Inc., OK, US) using Student's *t*-tests and Mann–Whitney *U*-tests. Results are expressed as mean ± STD. A *P* value less than 0.05 was considered statistically significant.

Results

Table 1 shows the demographic characteristics of normal subjects and patients with hemorrhoids. There was no statistical difference for age and gender between these two groups. Table 2 showed the SUV_{max} in normal subjects and patients with hemorrhoids. The SUV_{max} ranged from 1.8 to 4.1 with a mean value of 2.8 for normal subjects and ranged from 1.4 to 8.3 with a mean value of 2.9 for patients with hemorrhoids (Fig. 1). There was no statistical difference of the SUV_{max} between these two groups using Student's *t* tests. In the hemorrhoid group, the mean SUV_{max} was 2.7 for patients with external hemorrhoids, 2.9 for patients with internal hemorrhoids, and 2.7 for patients with mixed type of hemorrhoids. No statistical differences were noted among these three groups using Mann–Whitney *U*-tests.

The highest SUV_{max} in the normal subjects was 4.1. Of the 156 subjects with hemorrhoids, eight (5.1%) subjects had SUV_{max} greater than 4.1 (Fig. 2).

Table 1. Demographic characteristics of normal subjects and patients with hemorrhoids

	No.	Age	Sex	
		Mean ± STD	Male	Female
Normal	20	53.7 ± 11.2	11	9
Hemorrhoid	156	53.2 ± 9.6	94	62
External	3	51 ± 2.6	0	3
Internal	149	53 ± 9.6	92	57
Mixed	4	60.2 ± 12.7	2	2

Table 2. The FDG uptake (SUV_{max}) in normal subjects and patients with hemorrhoids

	No.	SUV_{max}		
		Lowest	Highest	Mean ± STD
Normal	20	1.8	4.1	2.8 ± 0.6
Hemorrhoid	156	1.4	8.3	2.9 ± 0.8
External	3	1.9	3.9	2.7 ± 1.1
Internal	149	1.4	8.3	2.9 ± 0.8
Mixed	4	1.8	3.5	2.7 ± 0.7

Discussion

Hemorrhoids are present in healthy individuals. The main observations of hemorrhoids are hemorrhage, thrombosis, and prolapse. Several theories have been proposed for the mechanisms of hemorrhoidal development including the varicose vein theory, the sliding anallining theory and the vascular hyperplasia theory, and the neovascularization theory [8, 9]. These pathological changes result in prolapsed anovascular cushions, which subsequently interfere with venous return. Decreased venous return, thought to be the mechanism of action, induces dilation of the venous plexus, venous stasis, and/or thrombosis [1]. Thrombosis in the venous plexus may elicit an inflammatory response. Moreover, vascular proliferation has been reported to cause an important pathologic change in hemorrhoids, which might be due to thrombosis formation [1]. Therefore, thrombosis, inflammation, and vascular proliferation should be considered in the pathogenesis of hemorrhoids.

In our study, eight (5.1%) subjects with hemorrhoids had SUV_{max} higher than 4.1, which was the highest

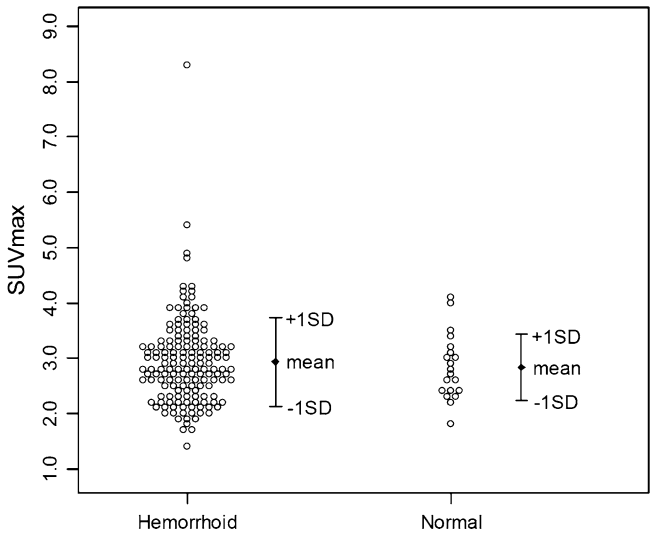

Fig. 1. Scattergram of SUV_{max} in 20 normal subjects and 156 subjects with hemorrhoids. The *solid lines* represent the highest SUV_{max} (4.1) in normal subjects.

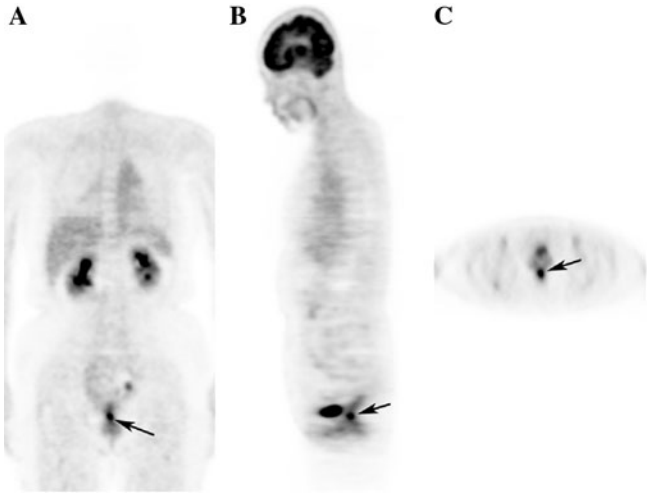

Fig. 2. The FDG-PET images show an area of increased FDG uptake in the anal region (*arrows*). **A** coronal view, **B** sagittal view, and **C** transverse view. The maximum SUV of the lesion is 8.3. A sigmoidoscopy was performed and internal hemorrhoids of the rectum was the final diagnosis.

SUV_{max} for the anal region in the normal group. The highest SUV_{max} in the hemorrhoid was 8.3 (Fig. 1), which was similar to the case reported by Lu et al. In her report, the SUV_{max} in the hemorrhoids was 8.0 [7]. In Basu et al.'s study, the intensity of FDG uptake in the hemorroids was also quite high (higher than that of liver uptake) although no SUV_{max} of the lesion was mentioned.

The exact cause of high FDG uptake in the hemorrhoids remains unclear. We considered inflammation, thrombosis, and vascular proliferation in the pathogenesis of hemorrhoids could have contributed to the high FDG uptake. It is well known that FDG not only accumulates in malignant tissue but also in pyogenic infectious foci, granulomatous and non-infectious inflammatory diseases [2]. Furthermore, FDG–PET has been used for the diagnosis and follow-up of large vessel vasculitis with good sensitivity (77%–92%) and high specificity (89%–100%) [3, 10–13]. FDG–PET has also been reported to be useful for the evaluation of thrombosis. In 2004, Kikuchi et al. [14] found marked accumulation of FDG in internal and external jugular vein thrombosis. In addition, Miceli et al. [15] reported that FDG–PET may be useful in evaluating response to treatment. In 2010, Khosa et al. [16] reported an increased FDG uptake not only within the vein but also the thrombus itself and concluded FDG–PET/CT appropriately shows venous thrombosis and might play a prominent role in the future. Recently, the relationship between angiogenesis and FDG uptake has been discussed [17, 18]. In an animal study, Calcagno et al. found a positive correlation between neovessel count in atherosclerotic plaques and ^{18}F-FDG uptake. They concluded that FDG–PET could be used as a clinical tool in the evaluation of lesion prognosis and monitoring of anti-angiogenic therapies [17]. Strauss et al. investigated patients with primary colorectal tumors with FDG–PET before surgery. Tissue specimens were obtained from the tumor during surgery, and gene expression was assessed using gene arrays. They found that FDG kinetics are modulated by angiogenesis-related genes. The transport rate for FDG is higher in tumors with a higher expression of VEGF-A and angiopoietin-2. The regression functions for the PET parameters provide the possibility to predict gene expression of VEGF-A and angiopoietin-2. They concluded that angiogenesis is a determining parameter for FDG kinetics in primary colorectal tumors [18].

Physiological FDG uptake in the intestines is frequently observed [19]. Typical sites of FDG accumulation in the intestinal tract include the gastroesophageal junction, gastric fundus, and colon (cecum, proximal ascending colon, and the recto-sigmoid colon). In research by Soyka et al. [20], they reported physiological FDG uptake with an SUV_{max} of 2.6 in the small intestine, 2.3 in ascending colon, 1.8 in transverse colon, 1.9 in descending colon, and 2.8 in recto-sigmoid colon. Recto-sigmoid colon had the highest mean SUV_{max} for the intestines. In our study, the mean SUV_{max} of the anal region was 2.8, which was the same as the SUV_{max} of recto-sigmoid colon in Soyka's study. Of 20 normal subjects, 13 (65%) had SUV_{max} greater than 2.5 for the anal region, 6 (30%) greater than 3.0 and 2 (10%) greater than 3.5. The exact mechanism and cause of the intestinal FDG uptake are still unclear. However, smooth-muscle activity, sphincter activity, constipation, and the presence of lymphoid tissue may be the causes [21, 22].

In conclusion, hemorrhoids may be considered as one possible cause of focal high FDG uptake in the rectum.

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Conflict of interest. The authors have no conflict of interest.

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